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HYPOKALEMIC PERIODIC PARALYSIS

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ABSTRACT Hypokalemic paralysis is an uncommon disease and often missed in the outpatient emergency settings. Early diagnosis and management is very important for a good outcome. History of previous similar episodes, positive family history and precipitating factors may help in arriving at the diagnosis. We are reporting a case of recurrent hypokalemic periodic paralysis presenting as quadriparesis in a young girl who was presented to our tertiary care hospital.

KEYWORDS :

INTRODUCTION :

Hypokalemic periodic paralysis is a disorder of muscle whereby voltage-gated ion channels (typically calcium or sodium, and less frequently potassium) are mutated, resulting in abnormalities of sarcolemma excitation. The disease typically first manifests in adolescence as bouts of mild to severe muscle weakness lasting hours and sometimes days associated with hypokalemia triggered most commonly by rest after exercise or high carbohydrate meals. Weakness typically recovers when serum potassium normalizes.[1]
Acute onset of quadriparesis is often due to neuropathic or myopathic disorders. The neurological causes of quadriparesis include traumatic diseases of spinal cord, acute transverse myelitis and demyelinating disorders. Polymyositis and metabolic muscle disorders can present as acute onset quadriparesis. Among metabolic muscle disorders, which include hypo and hypercalcemia, hypo and hyperkalemia, hypokalemic paralysis is the commonest cause. [2]

CASE REPORT:

A 23 years old female has presented on 19.08.2016 to our outpatient clinic with the H/o weakness of all four limbs for past 24 hrs. The weakness was acute in onset and non-progressive. There is no H/o cranial nerve involvement or sphincter disturbance. There is no pain or paresthesia in the limbs. There is no h/o fever, recent vaccinations, vomiting or diarrhea or any new drug intake. Patient gave the h/o similar illness earlier in the last 2 years and had been hospitalized, treated and improved on both occasions. The family history is suggestive of similar illness in the parent.

On examination, the patient found to be comfortable at rest, not dyspnoeic, no tachypnea, with a pulse rate of 86 per minute and BP of 100/70mmHG. Cardiovascular, respiratory and abdominal examinations are normal. Patient was fully conscious with normal higher functions of cranial nerves. Patient had a flaccid weakness of all four limbs with a power of 2/5 proximally and 3/5 distally. The patient also had a neck muscles weakness in the form of difficulty in lifting the head from bed. There is hypotonia of the muscles of upper and lower limb with absent deep tendon reflexes. The plantar was flexor on both sides. There is no sensory loss or sphincter disturbance.

On blood investigation at the time of admission revealed hemoglobin of 9.9 gms , total count of 10900 with platelet of 3.14 lakhs, Blood sugar, renal function and liver function tests were essentially normal. Serum electrolytes are 19/08/2016 showed serum sodium 143mEq/L, potassium 2mEq/L, Chloride 98mEq/L. Serial evaluation of serum electrolytes showed a potassium level of 2.7mEq/L, 3.3mEq/L &3.8mEq/L. Thyroid functions are normal. Serum magnesium 3.1mEq/L, calcium 10.1mEq/L ,phosphorous 3.4mEq/L. ECG shows a sinus rhythm with flat T waves in the anterior leads with normal PR interval, Patient was managed with inj. Potassium chloride 20mmol/3 hours and repeated according to the serum potassium level and later supplemented with oral potassium. Patient clinically started improving from the third day onwards and regained full power on day 5. Serum potassium level became stable and the patient was discharged on 26/08/2016 with an advice to restrict salt and high carbohydrate diet and Tab. Acetazolamide 250mg BD and to review after two weeks.

Periodic paralysis may be primary or secondary type. The paralytic attack can last from an hour to several days and the weakness may be generalized or localized.[3] Disturbances of potassium equilibrium can produce a wide range of disorders including myopathy, marked muscle wasting, diminution of muscle tone, power, and reflexes.[4] The primary hypokalemic periodic paralysis is autosomal dominant and is exacerbated by strenuous exercise, high carbohydrate diet, cold and excitement.[3] In the primary type, episodes of weakness recur frequently.

Familial hypokalemic periodic paralysis is an autosomal dominant inherited disease and is uncommon in female. The classic pathological changes include degeneration of muscle fiber with vacuoles filled with clear fluids. Though we have not done a muscle biopsy in this case, the positive family history, recurrent episode, clinical features and the improvement with potassium supplement all suggest hypokalemic periodic paralysis.

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